acquiesced to that point in a reply to the first of several letters to the editor by Brondum (2007) on the same topic. However, Braun et al. (2007) noted that including such information is not always possible when, for example, the use of National Health and Nutrition Examination Survey (NHANES) data or other constraints on study design do not allow it. Although we agree that failure to control for parental psychopathology is a weakness in many of the published studies reporting an association between childhood lead exposure and a diagnosis of ADHD, we believe that the consistency of the association across several published studies using different study designs adds to the weight of evidence that this is a real association and not a spurious effect due to uncontrolled confounding.

We hope that this reply clarifies the goals of our review for those faced with the challenge of assessing the neurobehavioral effects of emerging contaminants and their possible contribution to the phenotypic expression of ADHD or other neurodevelopmental disorders.

The authors declare they have no actual or potential competing financial interests.

Paul A. Eubig Andréa Aguiar Susan L. Schantz

Department of Comparative Biosciences College of Veterinary Medicine University of Illinois at Urbana-Champaign Urbana, Illinois E-mail: eubig@illinois.edu

REFERENCES

Braun JM, Lanphear BP, Kahn RS, Froehlich T, Auinger P. 2007. ADHD: Braun et al. Respond [Letter]. Environ Health Perspect 115:A399. .

Brondum J. 2007. Environmental Exposures and ADHD [Letter]. Environ Health Perspect 115:A398.

Eubig PA, Aguiar A, Schantz SL. 2010. Lead and PCBs as risk factors for attention deficit/hyperactivity disorder. Environ Health Perspect 118:1654–1666.

Home Energy-Efficiency Retrofits

doi:10.1289/ehp.1103621

In the February 2011 issue of *EHP*, Manuel (2011) took an important look at some potential adverse health implications of home energy retrofits. Here, we further discuss the complexity of possible indoor environmental concerns and encourage incorporation of comprehensive homeowner education campaigns in weatherization programs.

The reduction of air infiltration by air sealing is a common energy retrofit measure (McCold et al. 2008). Several field studies of weatherized homes have reported average reductions in air leakage of 13–40% (Berry 1997; Judkoff et al. 1988), although the impact of weatherization on actual air

exchange rates and indoor pollutant concentrations is poorly understood. Moreover, studies have seldom evaluated the effects of weatherization on low-income groups or vulnerable populations (e.g., asthmatic or elderly), although occupants in low-income residences are at higher risk for many indoor environmental hazards (Evans and Kantrowitz 2002), and some population subgroups may also be disproportionately affected by indoor air pollution (Hun et al. 2009).

Although some research exists on the impact of weatherization on indoor concentrations of combustion products, radon, and moisture, other indoor pollutants deserve attention. For example, Logue et al. (2011) identified nine priority indoor air pollutant hazards in U.S. residences, which, among others, have been associated with a wide range of both chronic and acute health effects (e.g., benzene, 1,4-dichlorobenzene, formaldehyde, naphthalene, particulate matter < 2.5 μm in aerodynamic diameter). Moreover, reducing air exchange rates in residences will likely increase indoor concentrations of reactive pollutants and the probability of chemical reactions occurring between them indoors (Weschler and Shields 2000), generating by-products associated with respiratory symptoms and asthma, such as low-molecularweight aldehydes, dicarbonyls, and secondary organic aerosols (Jarvis et al. 2005). On the other hand, reductions in air infiltration should decrease penetration of outdoor pollutants, which is of particular importance in traditionally leakier low-income households (Chan et al. 2005) in neighborhoods with high outdoor air pollution. Thus, we urge the environmental health community to investigate the net effects of weatherization on a wide variety of indoor and outdoor pollutants and health outcomes.

Implementation of home energy retrofits also creates an opportunity to incorporate innovative, engaging homeowner education strategies to reduce both energy consumption and indoor environmental risks. Occupant behavior has a major influence on both energy consumption (Allcott and Mullainathan 2010) and indoor exposures to pollutants (Meng et al. 2005). Furthermore, many indoor air quality risks can be mitigated with relatively simple home behavior practices, such as using exhaust fans, avoiding toxic cleaning chemicals, and using appropriate air cleaners (Brugge et al. 2003). However, we have learned from research on household energy consumption that educational materials alone usually fail to alter behaviors (Charles 2009). Greater energy savings from home retrofits could be achieved by complementing homeowner education campaigns with regular feedback on energy use and economically motivational programs (Peschiera et al. 2010). Additionally, home walkthroughs with trained building specialists can identify energy-inefficient behaviors and appliances in conjunction with potential indoor environmental hazards. These and other behavior-change strategies to promote green and healthy housing should be made available to weatherization programs across the country, and their effectiveness should be assessed. Because home weatherization is currently a priority of the federal government, this is a crucial time to address these fundamental research questions and implement the findings nationwide.

All authors are current or former trainees or affiliates of a National Science Foundation IGERT program in Indoor Environmental Science and Engineering at The University of Texas at Austin (award DGE 0549428).

M.C.J. is employed by Lennox International, Inc., which produces HVAC systems and products. The other authors declare they have no actual or potential competing financial interests.

Brent Stephens Ellison M. Carter Elliott T. Gall C. Matt Earnest Elizabeth A. Walsh

National Science Foundation Integrative Graduate Education and Research Traineeship (IGERT) Program in Indoor Environmental Science and Engineering The University of Texas at Austin Austin, Texas

E-mail: stephens.brent@mail.utexas.edu

Diana E. Hun

Oak Ridge National Laboratory Oak Ridge, Tennessee

> Mark C. Jackson Lennox International Inc. Carollton, Texas

REFERENCES

Allcott H, Mullainathan S. 2010. Behavior and energy policy. Science 327(5970):1204–1205.

Berry LG, Brown MA. 1994. Patterns of Impact in the Weatherization Assistance Program: A Closer Look. ORNL/CON-331. Oak Ridge, TN:Oak Ridge National Laboratory. Available: http://weatherization.ornl.gov/pdfs/ORNL_CON-331.

Brugge D, Vallarino J, Ascolillo L, Osgood ND, Steinbach S, Spengler J. 2003. Comparison of multiple environmental factors for asthmatic children in public housing. Indoor Air 13(1):18–27.

Chan W, Nazaroff W, Price P, Sohn M, Gadgil A. 2005. Analyzing a database of residential air leakage in the United States. Atmos Environ 39(19):3445–3455.

Charles D. 2009. Leaping the efficiency gap. Science 325(5942):804-811.

Evans GW, Kantrowitz E. 2002. Socioeconomic status and health: the potential role of environmental risk exposure. Annu Rev Public Health 23(1):303–331.

Hun DE, Siegel JA, Morandi MT, Stock TH, Corsi RL. 2009. Cancer risk disparities between Hispanic and non-Hispanic white populations: the role of exposure to indoor air pollution. Environ Health Perspect 117:1925–1931.

Jarvis J, Seed M, Elton R, Sawyer L, Agius R. 2005. Relationship between chemical structure and the occupational asthma hazard of low molecular weight organic compounds. Occup Environ Med 62(4):243–250.

Judkoff R, Hancock E, Franconi E, Hanger R, Weiger J. 1988. Mobile Home Weatherization Measures: A Study of Their Effectiveness. Golden, CO:Solar Energy Research Institute. Available: http://www.nrel.gov/docs/legosti/old/3440.pdf [accessed 8 June 2011].

Logue JM, McKone TE, Sherman MH, Singer BC. 2011. Hazard assessment of chemical air contaminants measured in residences. Indoor Air 21:92–109.

Manuel J. 2011. Avoiding health pitfalls of home energyefficiency retrofits. Environ Health Perspect 119:76–79.

McCold L, Goeltz R, Ternes M, Berry L. 2008. Texas Field Experiment: Performance of the Weatherization Assistance Program in Hot-Climate, Low-Income Homes. ORNL/CON-499. Oak Ridge, TN:Oak Ridge National Laboratory. Available: http://weatherization.ornl.gov/ pdfs/ORNL_CON-499.pdf [accessed 8 June 2011].

Meng QY, Turpin BJ, Korn L, Weisel CP, Morandi M, Colome S, et al. 2005. Influence of ambient (outdoor) sources on residential indoor and personal PM_{2.5} concentrations: analyses of RIOPA data. J Expo Anal Environ Epidemiol 15(1):17–28.

Peschiera G, Taylor JE, Siegel JA. 2010. Response-relapse patterns of building occupant electricity consumption following exposure to personal, contextualized and occupant peer network utilization data. Energy and Buildings 42(8):1329–1336.

Weschler CJ, Shields HC. 2000. The Influence of ventilation on reactions among indoor pollutants: modeling and experimental observations. Indoor Air 10(2):92–100.

Environmental Health Research Implications of Methylmercury

doi:10.1289/ehp.1103580

In "Adverse Effects of Methylmercury: Environmental Health Research Implications," Grandjean et al. (2010) reviewed the scientific discoveries of health risks resulting from methylmercury exposure, including the history of the Minamata disease incident. Although their title states "research implications," the authors failed to convey some important caveats from the incident.

First, Grandjean et al. (2010) explained the incident as if serious delays of the recognition of "the exact cause (methylmercury)" deferred the corrective action. However, recognition of an etiologic agent is not a necessary condition for prevention (Goodman et al. 1990). When source and transmission are identified, they must be eliminated even if the etiologic agent is unknown. In the case of Minamata disease, even in 1956 when the first patient was identified, eating contaminated seafood was determined to be a cause of the disease; this occurred 3 years before the etiologic agent was identified (Tsuda et al. 2009). Grandjean et al. (2010) cited Harada (2004), who wrote,

However, with no specific causative substance [etiologic agent] determined, there was no legal basis for a ban on fishing. (Under Item 2 of the Food Sanitation Act, it was not possible to prohibit fishing while the cause was undetermined.)

However, in Japan, even with no specific etiologic agent determined, the Food Sanitation Act has routinely been enacted when causal food and/or causal facility was determined.

Second, Grandjean et al. (2010) mentioned the "diagnostic difficulties" of methylmercury poisoning cases. Lack of investigation of the Minamata disease incident as food poisoning resulted in unnecessary diagnostic difficulties; such difficulties do not usually arise in food-poisoning incidents in Japan. In the case of Minamata disease, in 1977 the Japanese Ministry of Environment (JME) established the criteria for diagnosis, which required combinations of signs that were advocated by the JME to be medically correct. However, the truth is that the JME recognized a lack of medical evidence on the criteria [Committee on Research and Human Rights/Japanese Society and Psychiatry and Neurology (CRHR-JSPN) 2003]. Moreover, medical researchers in Japan have pointed out that the criteria were medically incorrect (CRHR-JSPN 1998). The "diagnostic difficulties" may have obscured who was affected and had neurological signs.

Third, Grandjean et al. (2010) stated that, "Only in 2009 was a law enacted to provide compensation to most of the remaining group of victims." However, it was not compensation. For Minamata disease, unless the affected persons are diagnosed by the above-mentioned criteria, they are not counted as patients and are thus not properly compensated. About 2,200 patients have been diagnosed with Minamata disease and have been compensated, whereas at least several tens of thousands of victims who have neurological signs characteristic of methylmercury poisoning have not been recognized as patients and have not been not properly compensated (McCurry 2006).

Fourth, Grandjean et al. (2010) described the "scientific account" of the cat experiment in 1959, which was published after a 40-year delay (Eto et al. 2001). However, the report provided only pathological findings, and the detailed explanation of the cat experiment had already been published in 1965 (Tomita 1965). The latter would be enough for prevention and control.

Finally, because the JME and local governments have been defendants in Minamata disease lawsuits, research funds from JME and the local governments may affect researchers' attitudes, possibly causing conflicts of interest.

T.T. and M.H. have provided expert testimony on Minamata disease. The other author declares he has no actual or potential competing financial interests.

Toshihide Tsuda

Department of Environmental Epidemiology Okayama University Graduate School of Environmental Science Okayama, Japan E-mail: tsudatos@md.okayama-u.ac.jp

Takashi Yorifuji

Department of Epidemiology Okayama University Graduate School of Medicine, Dentistry and Pharmaceutical Sciences Okayama, Japan

Masazumi Harada

Department of Social Welfare Studies Kumamoto Gakuen University Kumamoto, Japan

REFERENCES

CRHR-JSPN (Committee on Research and Human Rights/ Japanese Society and Psychiatry and Neurology). 1998. An opinion on the criteria on acquired Minamata disease [in Japanese]. Psychiatr Neurol Jpn 100:765–790.

CRHR-JSPN (Committee on Research and Human Rights/ Japanese Society and Psychiatry and Neurology). 2003. An opinion about Minamata disease accreditation system and medical specialists' involvement [in Japanese]. Psychiatr Neurol Jpn105:809-834.

Eto K, Yasutake A, Nakano A, Akagi H, Tokunaga H, Kojima T. 2001. Reappraisal of the historic 1959 cat experiment in Minamata by the Chisso factory. Tohoku J Exp Med 1944):197-203.

Goodman RA, Buehler JW, Koplan JP. 1990. The epidemiologic field investigation: science and judgment in public health practice. Am J Epidemiol 132:91–96.

Grandjean P, Satoh H, Murata K, Eto K. 2010. Adverse effects of methylmercury: environmental health research implications. Environ Health Perspect 118:1137–1145.

Harada M. 2004. Minamata Disease (Sachie T, George TS, translators). Tokyo:lwanami Shoten.

McCurry J. 2006. Japan remembers Minamata. Lancet 367:99-100.

Tomita H. 1965. Minamata disease 12 [in Japanese]. Goka 7:346–353

Tsuda T, Yorifuji T, Takao S, Miyai M, Babazono A. 2009. Minamata disease: catastrophic poisoning due to a failed public health response. J Public Health Policy 30:54–67.

Methylmercury: Grandjean et al. Respond

doi:10.1289/ehp.1103580R

We thank Tsuda et al. for sharing their views. Along with their previous publications on Minamata disease, we find their comments useful as a complement to our brief historical review of the mass poisonings in Japan and associated events (Grandjean et al. 2010). However, the specific issues raised in their letter do not affect our conclusions on the research implications of the history of methylmercury science.

P.G. has provided paid expert testimony on mercury toxicology in a legal case concerning environmental pollution from coal-powered power plants. The other authors declare that they have no actual or potential competing financial interests.

Philippe Grandjean

Department of Environmental Health Harvard School of Public Health Boston, Massachusetts E-mail: pgrand@hsph.harvard.edu